J. MYLES BICKERTON

THE INHERITANCE OF BLINDNESS

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THE INHERITANCE OF BLINDNESS

The Contribution of Eugenics to the Reduction of Eye Disease

By J. MYLES BICKERTON, F.R.C.S.

(A paper read to the Eugenics Society on March 15th, 1932)

THE total world figures relating to blindness must be of staggering dimensions. In China, one disease (trachoma) has been estimated to have blinded one million in both eyes, 3 to 4 million in one eye, and to have gravely injured 20 million—this out of a population of, say, 400,000,000.

In the United Kingdom the registered blind were reported as follows :

TABLE 1

Year.	England and Wales.	Scotland.	Total.
1919	25,840	—	—
1929	52,727	—	—
1930	56,853	8,516	65,369
1931*	62,727	10,000	72,727

Around 8,000 of these blind are also mentally defective, and many are deaf.

There has been an increase, therefore, of 35,000 odd in the registered blind in the last twelve years, while that for the last year was 4,126—i.e. over eleven a day. The total figure, however, is by no means a true indication of our blind population, for the following groups of persons are largely excluded, and may increase it to *250,000 blind persons* :

1. Almost all blind persons of the well-to-do classes.
2. Infants below school age (many die blind).
3. Those persons who become blind after leaving school at fourteen years or later, until reaching blind pension age at fifty years. Many of these persons refuse to be certified, regarding it as rather a disgrace.
4. Those persons who become blind after 65 years of age are often not registered. They obtain the old-age pension, which is the same in amount as the blind pension, 10s. weekly. This is a very large group, and is likely to grow as the average length of life increases.
5. Many blind persons of a suitable age to obtain the blind pension, between the ages of 50 and 65 years, do not apply for it because they have never heard about it.

The certification of the blind, however, is done in a very haphazard way in this country, and the real causation of blindness is not truly shown, so that the figures in Table 2 are only approximate. A child, I should explain, is considered blind if it cannot read school books with safety to the eyes ; while the criterion for an adult is the capacity to do work for which eyesight is necessary.

I have recently been on a sub-committee which has made strong recommendations to the Ministry of Health to improve the registration, and if this is done, heavy blind figures will very shortly be forthcoming.

A glance at Table 2 will show that blindness in the more active years of life is very largely avoidable, while much of it is due either to definitely dysgenic births or to the unrestricted production of children in poor or bad circumstances. In this table I have given the groups of the blind and the percentage in each group which could, in my opinion, be greatly reduced by the prevention of birth—since for practical purposes bad genes and certain types of bad environment may be included together as causes of

* The figures for 1931 are estimates.

TABLE 2.—*Rough Figures of Causation of Blindness in the United Kingdom.**

Disease.	Per cent.	Largely avoidable per cent.	No. of blind at 100,000.	No. of blind at 250,000.
Trachoma	2	2	2,000	5,000
Purulent Conjunctivitis	2	1	2,000	5,000
Ophthalmia Neonatorum	5	5	5,000	12,500
Accident	5	2	5,000	12,500
Congenital Defects	6	6	6,000	15,000
Senile Degeneration	10	—	10,000	25,000
Glaucoma	10	1	10,000	25,000
Myopia	14	14	14,000	35,000
Syphilis	15	10	15,000	37,500
Senile Cataract	15	1	15,000	37,500
Others	16	2	16,000	40,000
	100	43	43,000	107,500

The 43 per cent. in column 2 may represent 70 per cent. of blind years lived.
Those blind in one eye may number about 1,000,000.

*Under one year of age.**

Cause.	Per cent.	Category.
Accident	2	neglect.
Syphilis	5	heredity.
Purulent Conjunctivitis	8	dirt.
Congenital Defects	30	heredity.
Ophthalmia Neonatorum	50	dirt.
	100	

(21 per cent. of the blind are blinded in the first year of life.)

* Column 1 is modified from Harman, and the other figures are my own estimates.

blindness which can only be eliminated by preventing the birth of sufferers.

TRACHOMA, 2 per cent.—This disease does not occur in clean households (see China, Egypt, etc.).

PURULENT CONJUNCTIVITIS, 2 per cent.—This condition rarely occurs in clean households, and I reckon that 1 per cent. is entirely avoidable (only one child in five is born into a clean home in England).

OPHTHALMIA NEONATORUM, 5 per cent.—This disease, due to gonococcal infection at birth, could and should be entirely wiped out by the prevention of unsuitable births.

ACCIDENTS, 5 per cent.—A fair number of these occur to infants, a fact which has considerable relevance to the limitation of births. A woman explained in a South London court, for instance, that she was the only person alive and responsible for 68 children (made up by three marriages; 26 of her own children, 22 of her second husband's, and 20 of her last husband's, all other parents being dead). There was recently reported a family in Buckinghamshire of 39 children, and one woman, newly married, had nine children in 2½ years. How can she manage, with a husband earning £3 a week?

SYPHILIS, 15 per cent.—I reckon that 10 per cent. of the syphilitic blind owe their condition to congenital syphilis, which might be called a 'pseudo-heredity'—since it never goes beyond the second generation

—and which could be entirely eliminated by preventing syphilitic women from having children.

Naturally, if a woman has been shown (by the Schick, Wassermann tests, etc.) to be syphilitic, she should avoid conception at all costs, unless and until she has been completely cured. If, however, her condition is not discovered until after conception, the problem becomes more difficult; but treatment during pregnancy will always obtain a non-syphilitic child if the treatment is thorough and commenced around the fourth month. If treatment cannot be thorough the choice is between the birth of a congenital syphilitic and inducing an abortion; and I, for my part—though I know many will not agree with me — am strongly inclined towards the latter.

Congenital syphilis, which shows itself in the eyes anywhere between 2 and 35 (usually 5 to 25) years of age, is a slow and terribly distressing affliction to patient, parents, and even doctors. From two to ten years' treatment is often necessary before even a quiescent stage is reached, and it may cause blindness in three different ways —by interstitial keratitis (or inflammation) of the cornea, by a widespread choroiditis with destruction of the retina, or by juvenile optic atrophy. Needless to say, too, a congenital syphilitic is often an ament, and is never really healthy.

In my opinion, therefore, Wassermann (or other) tests should be compulsory in appropriate cases, and a positive indication should be followed by treatment, contraception, sterilization, or abortion, according to the circumstances. I have said "compulsory," but I believe that if the women of England knew that congenital syphilis, with all its terrible concomitants, could be entirely eliminated, they would welcome the appropriate routine from the medical profession. As it is, large numbers of syphilitics marry and produce defective children; and at King's College Hospital last year 34 per cent. of the new syphilis cases—mostly eye—were congenital.

My chief concern here, however, is with those types of blindness which are strictly hereditary—glaucoma (1 per cent.), extreme myopia (14 per cent.), senile cataract (1 per cent.), and other types (2 per cent.)—a total of 24 per cent., or practically one quarter of all forms of blindness. As a prelude to discussing them, I should like to repeat the platitude, too often forgotten, that normal sight (emmetropia) is also hereditary, a great number of genes being jointly at work in its production. The various departures from normality are due to the aberration of one or more members of this gene-complex.

GENETIC TYPES OF BLINDNESS

Heredity is the study of differences or variations, the laws of which show that we may all differ in three ways:

1. By modification (environment).
2. By combination in accordance with Mendel's laws.
3. By mutation, the production of new hereditary types.

In certain simple forms of life 10 per cent. of the offspring are mutations; and according to Darwin's law of natural selection, the best or hardiest of these varieties survive. Thus variation has a beneficial effect on the community if the law of natural selection is operative. The trouble in England now is that we have dysgenic selection.

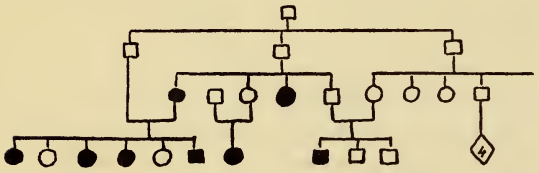
Mutations act *very well plus* eugenic selection. They act *fairly well plus* natural selection. They act *badly minus* natural selection, and *very badly plus* dysgenic selection.

We must get back to natural selection first, and then to eugenic selection. This conclusion is logically irrefutable. Education and environment have remarkable effects upon the individual during his lifetime, but they do not influence his hereditary equipment.

We know more about the hereditary diseases of the eye than about those of any other organ, and for the good reason that, being the most important and complicated of our sense organs, its slightest defects cause marked disturbances of function.

The genetics of variations in normal eye-colour are too well known—though not yet

finally elucidated—for mention here, and the abnormal condition of albinism is also fairly widely recognized as a simple recessive. But the eye defects associated with albinism are not so generally realized, nor is it always understood that they are really but symptoms of a general deficiency. Practically all albinos are below the average in health and vitality, many are aments, and all, as a direct result of their condition, are photo-phobic, because their eyes lack the pigment necessary to protect the sensitive retina. The consequence is that they hang their heads, pucker their brows, and half-close their eyelids to shut out the light; and often, moreover, they suffer from nystagmus or myopia. The incidence in the population is 1 in 10,000, and 30 per cent. of it is due—like so many similar recessives—to consanguineous marriages.



Albino (Tertsch).

Chart 1.

THE MYOPIA GROUP.—The genetics of this varied group have not been fully disentangled, but one can say for certain that short sight never arises in the absence of a hereditary predisposition, though that predisposition may remain latent in good health or be accentuated by illness and physical disability—as the myopia increases, the eye stretches until the retina is destroyed. Dysgenic births are the chief cause of severe myopia, and many high myopes go blind, while there are immense numbers whose very defective vision is practically equivalent to blindness, but who do not come within the scope of the Blind Persons Act.

Without intending to imply that it is a simple unit character, I must agree with Clausen, who considers that myopia tends to be recessive. Wilson, for instance, found that in 100 families with one myopic parent there were 200 myopic children and 250 non-myopic; while in 91 families with neither parent myopic, only 100 children were myopes and 300 non-myopes.

Therefore although short-sighted offspring are often born of normal parents, when both parents are short-sighted their children invariably suffer from the same defect.

Sex-linked Myopia (Worth).

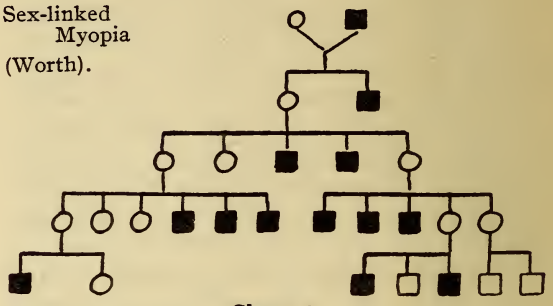


Chart 2.

Dominant Myopia, with nystagmus (Vogt).

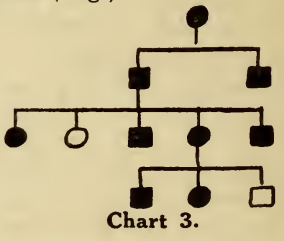


Chart 3.

Wilson's other figures are interesting.

In 677 myopic children, hereditary evidence was as follows:

Grandparents	54
Mothers	369
Fathers	254
Siblings	164
Uncles, aunts	31
Cousins	9
Father to son	79
Father to daughter ...	149
Mother to son	111
Mother to daughter ...	231

Hereditary evidence was found in:

Low myopia,	up to 3 dioptres	65 per cent.
Medium,	4-6 dioptres	66 per cent.
High,	7-12 dioptres	67 per cent.
Very high,	over 12 dioptres	67 per cent.

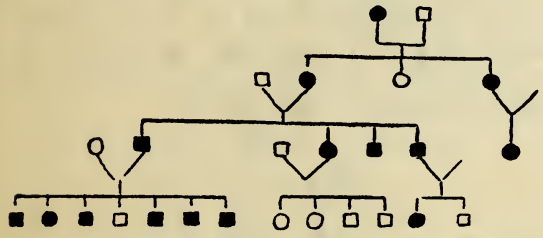
Hereditary evidence was found in the following occupations:

School children	... 65 per cent.
Home workers	... 63 per cent.
Factory workers	... 68 per cent.
Clerks ...	... 60 per cent.
Labourers	... 60 per cent.

The run of the figures in these last groups indicates independence of environment.

Reference must be made here to DETACHMENT OF THE RETINA, which is a fairly common sequel to high myopia. Bogatch reported a family of eleven high myopes in seven of whom detachment of the retina supervened in one or both eyes. Retinal detachment is found as a dominant, recessive, or male sex-linked in different pedigrees.

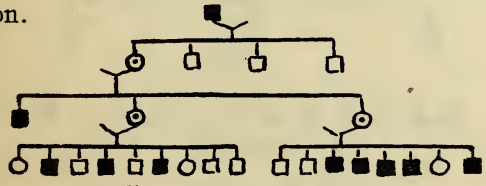
In the same way, ASTIGMATISM, LONG SIGHT, AND SQUINT (which is often associated with long sight), may be inherited. Squint, from which more than 2 per cent. of school children suffer, is responsible for a large number of blind eyes.



Long sight—Internal squint (Clausen and Bauer).

Chart 4.

NYSTAGMUS, PTOSIS, OPHTHALMOPLEGIA.—These may occur generation after generation.

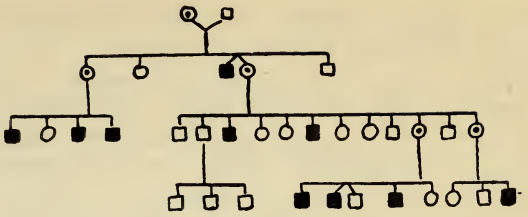


Hereditary Nystagmus (Holm).

Chart 5.

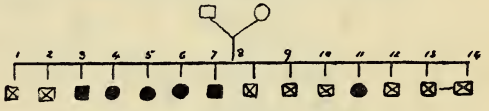
MICROPHTHALMIA, or very small eye, with anophthalmos (lack of an eye) as the extreme form, has been found as a dominant, recessive, or recessive sex-linked (Ash) (next column).

RETINITIS PIGMENTOSA.—Czellitzer says that nearly 4 per cent. of all the blind are due to hereditarily determined atrophy of the retina. Some 300 pedigrees are given in the *Treasury of Human Inheritance.* The condition begins in early youth and progresses to complete blindness sooner or later, often around forty years of age. As in other diseases, there are several forms, as shown by their differing heredity, the commonest being recessive—33 per cent. of those thus affected owe their condition to consanguinity. In some families the condition is linked with deafness.



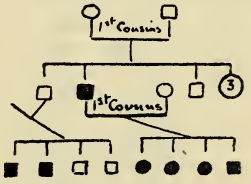
Microphthalmia (Ash).

Chart 6.



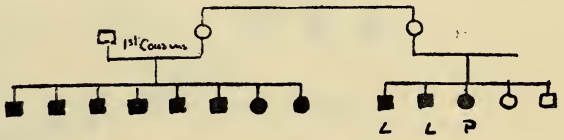
Retinitis Pigmentosa (Sambuc).

Chart 7.



Ret. Pigmentosa (Mooren).

Chart 8.



Ret. Pigmentosa (Mann). Leber's Optic Atrophy.

Chart 9.

OPTIC NERVE ATROPHY AND OPTIC NEURITIS.—These frequently cause blindness, and the hereditary variety is well recognized by ophthalmologists, 1,000 pedi-

grees being available for study. 85 per cent. of those affected are males. The disease comes on at different ages, the average being about twenty. Diminution of vision is the chief symptom and often appears suddenly; there is a central scotoma, and after six months or so the disease remains stationary. The malady has been traced once through six generations, twice through five generations, and often through four generations.

LEBER'S OPTIC ATROPHY is a name for the sex-linked variety. It appears to be an a-biotrophy, or lack of vital force, occurring at the prime of life.

Optic atrophy occurs also in another hereditary disease, FRIEDREICH'S ATAXIA, which is a severe recessive ataxia. It is very crippling, but the patients live to old age and are a great burden on their relatives (or on the State). A study of all cases shows that a common ancestor is probable; and it is by no means improbable that all those in Switzerland derive ultimately from a common source. It is certainly found most frequently in remote valleys where inbreeding has been common.

There is a dominant ataxia, cerebellar ataxia, or MARIE'S DISEASE.

ANIRIDIA (absence of the iris).—All such cases that I have seen have been of blind standard. I particularly wish to call attention to the Risley pedigree (1915) of aniridia. One blind man with aniridia, had

13 children similarly affected (100 per cent.),
61 grandchildren out of 63,
39 great-grandchildren out of 42—
114 blind out of 119 (Chart 14).

CONGENITAL CATARACTS AND SENILE CATARACTS (Charts 15-17). — These are usually dominant, and are very important, Macklin stating that 13 per cent. of the pupils in blind schools are blind through cataract, and Hirst giving 10 per cent. in 1,300 blind persons (anophthalmia and microphthalmia came next).

(Hawkes.) Hered. Optic Atrophy.

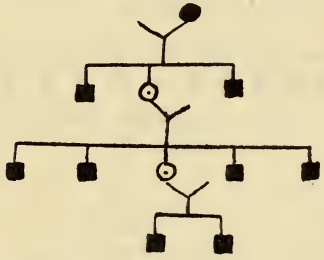


Chart 10a.

(Bickerton, 908.)

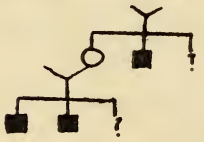


Chart 10b.

Hered. Optic Atrophy (Thomsen, 802).

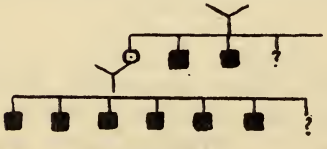


Chart 11.

Hered. Optic Atrophy (Norris, 750).

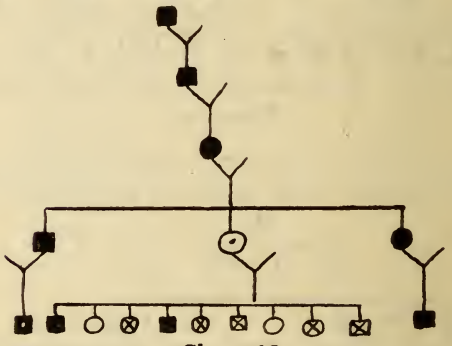


Chart 12.

(Norris, 881.)

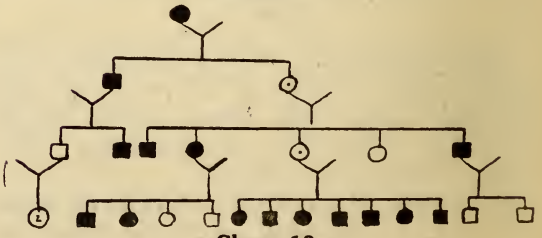


Chart 13a.

Hereditary Optic Atrophy (Hogg, 837).

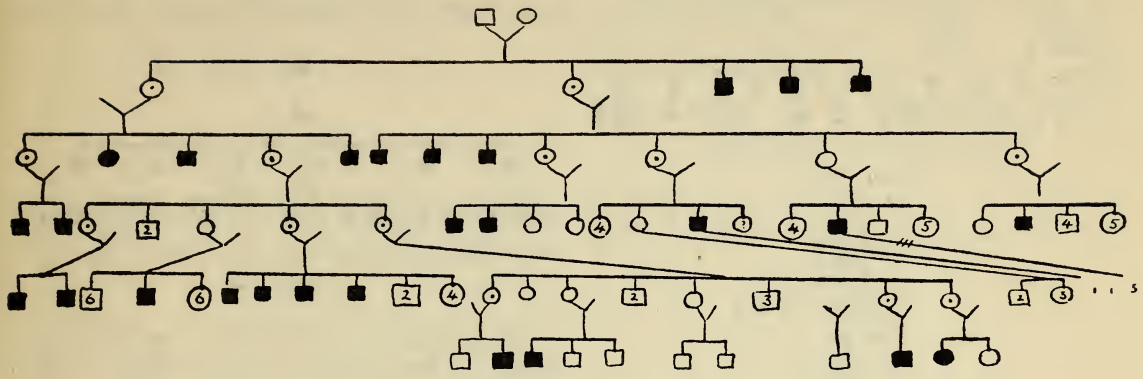
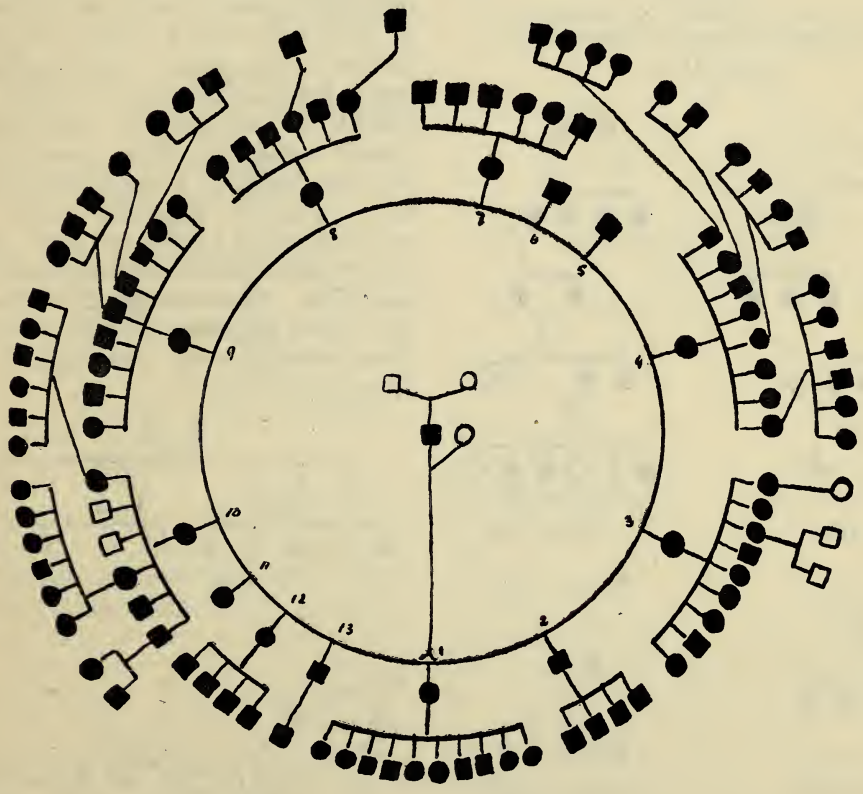


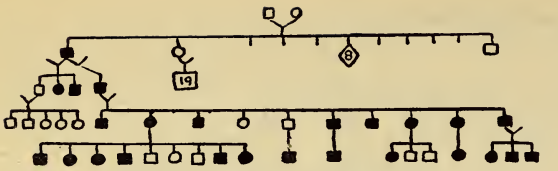
Chart 13b.

Aniridia [Absent Iris] (Risley, 1915).

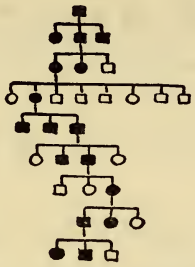


I
|
13 Affected children (all blind).
|
63-61 Defective (blind).
|
42-39 ,, ,,

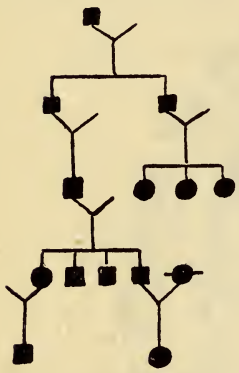
Chart 14.



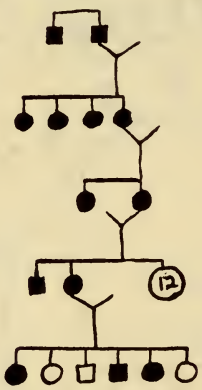
Lamellar Cataract (Nettleship).
Chart 15a.



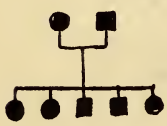
Dominant Night Blindness. Nougaret family. 2,116 persons; 135 affected (Nettleship).
Chart 15b.



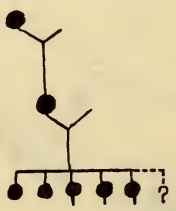
Cataract (Loeb, 328).
Chart 16a.



(Fromaget, 290 L.)
Chart 16b.



Cataract (Loeb).
Chart 17a.

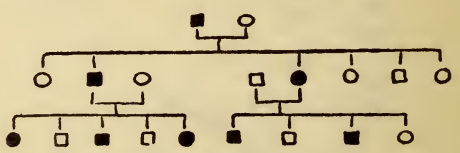


Coralliform Cataract (Nettleship).
Chart 17b.

ECTOPIA LENTIS (congenital displacement of the lens) has been traced as a dominant through six generations, though in some pedigrees it is recessive. Cameron's pedigree shows both lenses affected in each case.

Hereditary Dislocation of Lens (both) (Cameron).
Chart 18.

GLAUCOMA.—In this group the hereditary factor is not always very marked, though congenital glaucoma does occur, and there is a probable hereditary predisposition to it in many other cases. This is known as hereditary anaphylaxis, or abiosis—a lack of vital force which causes the structure to die before its time. Many pedigrees suggest a dominant heredity, but the hereditary course is occasionally interrupted, and casual causes are often necessary to bring out the disease. It may arise early in life as a juvenile glaucoma, and ostensible anticipation is known to occur, though this can be explained by unintentional statistical selection. Occasionally glaucoma is recessive, and appears then to be common in Jews, among whom consanguinity is fairly frequent.



Inflam. Glaucoma (Howe).
Chart 19.

Juvenile Glaucoma (Kamenetzki).
Chart 20.

Glaucoma (Bickerton, 1932).

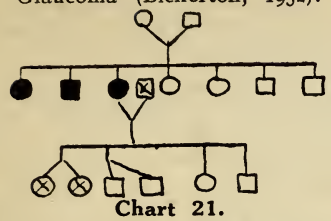


Chart 21.

BLUE SCLEROTICS.—This is a condition in which the coat of the eye is too thin or too transparent, accompanied by a failure in development of connective tissues. It is often associated with fragilitas ossium, or brittle bones, and with a type of deafness due to oto-sclerosis. The female is more likely both to show and to transmit it.

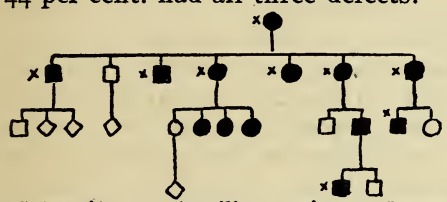
Seventy-three pedigrees since 1903 show 463 defective individuals; it is usually dominant.

Of adults with blue sclerotics:

60 per cent. had liability to fractured bones.

60 per cent. had oto-sclerosis.

44 per cent. had all three defects.



Blue Sclerotics. x Fragilitas ossium. (Burrows.)
Oto-sclerosis 40 per cent.

Chart 22.

GLIOMA.—This is a rare malignant growth, occurring in infants, of the nerve elements of the eye, which in some families shows a strong hereditary tendency, and is apparently recessive. It usually affects both eyes, and their early removal is necessary to save life. Some sufferers survive, marry, and often transmit the defect. Bell found 128 cases in 36 pedigrees; Clausen's sibships showed 83 affected and 61 unaffected. Mohr considers the condition to be lethal in a homozygous condition.

The death of a child with glioma of the retina is an appalling experience for parents and child, and may take as long as two years. The law which forces us to withhold euthanasia in these cases, and at the same time forces us to act in a barbarous and monstrously inhuman fashion, is, in my opinion, indefensible.

Glioma Retinæ (Newton, 339).

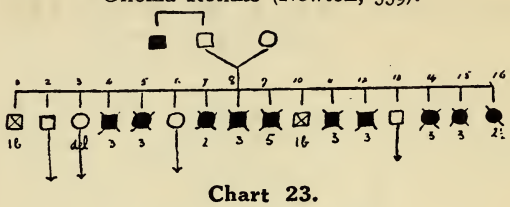


Chart 23.

Glioma (Wilson, 349).

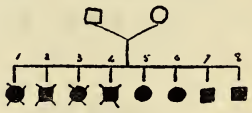


Chart 24.

Glioma (Thomson and Knapp, 335).

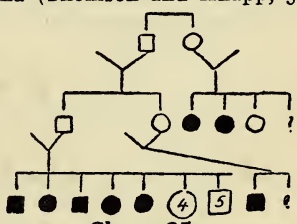


Chart 25.

Glioma (Griffith, 333).

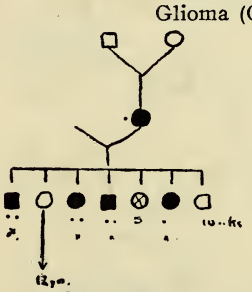


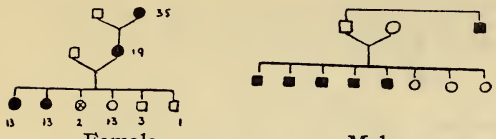
Chart 26a.

(Letchworth, 1898.)

Chart 26b.

MACULO-CEREBRAL DEGENERATIONS.—Several conditions might be described under this heading, including the rare disease of the central nervous system known as amaurotic family idiocy. Commencing at about five or seven years of age, the children go blind within two years and mentally degenerate within the next two; death occurs around 17-18 years. Amaurotic family dementia is a diffuse tapeto-retinal degeneration causing total, or nearly total, blindness, beginning at birth or later, and

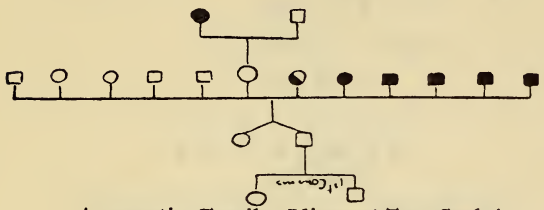
frequently associated with cerebral degeneration producing idiocy. It is a not infrequent cause of congenital blindness.



Female. Male.

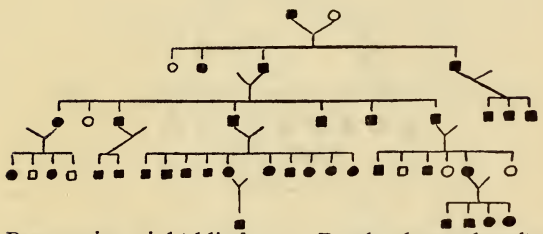
Amaurosis Sex-linked (Sedgwick).

Chart 27a. **Chart 27b.**



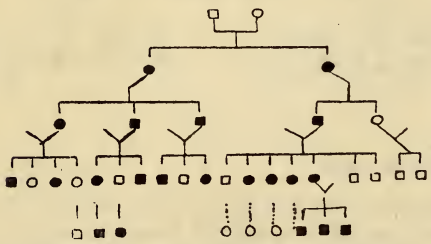
Amaurotic Family Idiocy—'Tay Sach' (King, Rus, Stewart).

Chart 28.



Progressive night-blindness. Death 16 months after complete blindness (Bordley).

Chart 29.

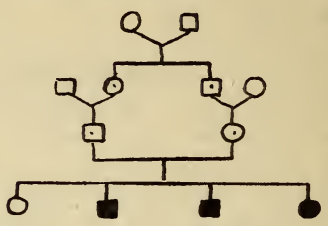


Stationary night-blindness (Sedan).

Chart 30.

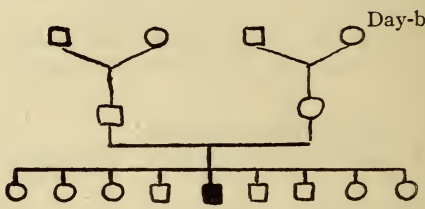
NIGHT BLINDNESS (above).—This is due to a defect of the rod vision (colourless vision). At dusk these cases are blind, though their sight is normal in good daylight. The famous Nougaret pedigree, by Nettleship, shows 135 affected persons out of 2,116 in nine generations (a dominant heredity). There are also a recessive sex-linked type, which is regularly associated with myopia, and one or more simple recessive types (Switzerland, Japan).

DAY BLINDNESS.—A cone vision defect. These cases are colour-blind and also of "blind standard."



Day-blind (Hessberg).

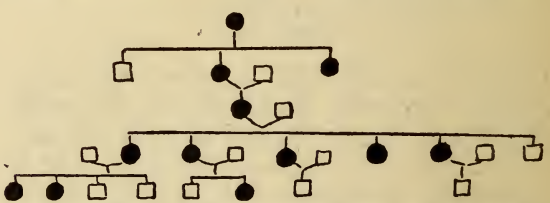
Chart 31.



Day-blind (Lutz).

Chart 32.

COLOUR-BLINDNESS.—This is usually a well-defined, recessive, sex-linked character, which affects 5.8 per cent. of males and .5 per cent. of females; but there seems also to be a rarer form of a recessive nature. Any such defect in colour vision, of course, incapacitates the affected individuals for work as railwaymen, sailors, motorists, etc.



Colour-blind. (Dominant Female Sex-linked.) (Rare form.)

Chart 33.

SOME SUGGESTED REMEDIES

Crude though effective methods for the relief of suffering have been practised in many parts of the world. A Chinese potentate, for instance, the ruler of a province, whose soul was torn by the sufferings of the lepers in his State, consulted with his officials. It was decided that he should call

together all the lepers from far and wide, to a banquet where amidst beautiful flowers and music, and plied with unlimited quantities of food and wine, they soon became oblivious to life and to their infirmities. At this stage the banqueting hall was set on fire, and next day all was lovely once more, pain and suffering had been banished from his State. This is a true story. The Esquimaux eliminate unwanted children. In Brazil, surplus children are sold into service without undue hardship to them or their parents; but in England, on the coldest night for three years (in February 1932), a child was left on a doorstep in Esher, Surrey, near the richest city in the world.

Our problem to-day is how to make the essential humanity of these principles conform to the sentiment of our time. Fortunately, our knowledge of the laws of biology—which are just as much the laws of God as those which govern the motions of the planets—enables us to reduce hereditary suffering just as effectively as did the more drastic methods of other times and countries.

A racial defect can be eliminated just as thoroughly by the sterility of its bearers as by their death; and I am therefore in the fullest sympathy with this *Society's* campaign to prevent the procreation of aments and the social problem group.

My concern in this article, however, is with the hereditary blind who, indeed, present an easier problem. They should not only be allowed, but encouraged, to marry, but on the one condition that by birth control, sterilization, or abortion, they avoid having children. I am aware that many members of this *Society* will disapprove of my advocacy of the last method, and it is only my own opinion that it is suitable for certain cases. I also consider that the law should not force a pregnant woman, as it does at present, to have a child against her wish—especially as this law is no more effective than those against the sale of sweepstake tickets in England or alcohol in the U.S.A.

No woman with active syphilis should be allowed to have a child.

Euthanasia for infants with gross defects might be available for parents who wish to make use of it—though here, again, I realize that all eugenists may not agree with me.

The other reforms I most wish to see are exactly those advocated by the *Eugenics Society*—the teaching of biology to all children and young persons, the formation of biological health centres or of national *constructive* birth-control clinics, and the active co-operation of the medical profession in the eugenics movement, which should be looked upon as a form of preventive medicine.

WHAT HAS—AND MIGHT BE—DONE

I have here indicated some—among many—methods which would prevent much needless suffering. Those who think that there is something to be said for action along these lines, but who fear lest public opinion may be difficult to move, may remember the past fights—some of them very recent—which have been won.

1. The washing of the body was regarded by devout Christians as a heathen custom, dangerous to the believer, in the time of Philip II of Spain, who had all the Moorish baths destroyed.

2. About the year 1780, Hanway invented the umbrella, which was considered by many as an unjustifiable interference with the laws of God.

3. My father-in-law tells me he remembers young children, both girls and boys, being forced up chimneys to sweep them. Some were choked and suffocated and others roasted; but Bill after Bill was rejected by our eminent gentlemen in Parliament, and it was not until the jointed brush was invented that a stop was put to this practice.

4. Bills brought before Parliament for the prevention of animal torture at fairs, and at times of public holidays, were greeted with laughter and ridicule, and the practice did not cease until 1830 or thereabouts.

5. Bills for the prevention of cruelty to children were opposed on the grounds that

they were seeking to interfere unwarrantably with the rights of parents. At this time the frightful conditions of child labour were considered unavoidable.

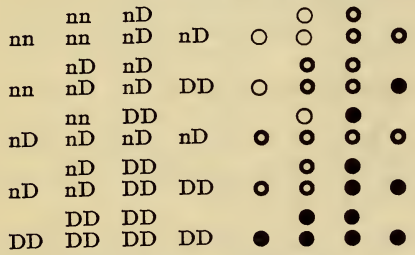
6. Hickman, who originated experiments in anæsthesia about 1822, was refused permission to read a paper before the Royal Society when Sir Humphry Davy was President. When anæsthesia was first introduced to relieve the pangs of childbirth, the pulpits, even up to 1847, resounded with denunciations of this practice on the grounds that this was interfering with the punishment of woman for her share in the original sin.

7. When Darwin's *Origin of Species* wa published, the religious opposition wa frantic.

The present prospects of the eugeni movement are certainly far better than wer those of the reforms I have mentioned especially since the practical proposals sug gested here and by others are fully in accor with the sentiment of the day. They woul reduce much suffering and cause none, an should therefore especially appeal to th religious bodies of this country—who have now a finer opportunity than ever before o capturing the imagination of the people an of guiding the ethics of the community.

THE SIMPLER MODES OF INHERITANCE

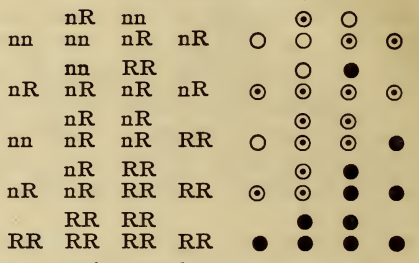
1. *Dominant Heredity*



nn homozygotic normal
nD heterozygotic defective
DD homozygotic defective

Cataract, congenital
Microphthalmos
Myopia
Blue Sclerotics
Ectopia Lentis
Aniridia
Ptosis, Nystagmus
Congenital Night Blindness
Glaucoma, congenital
Retinal and Optic Atrophy
Reticular Corneal Opacity
Nodular Corneal Opacity
Buphthalmos

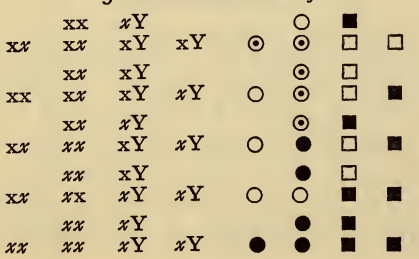
2. *Recessive Heredity*



nn homozygotic normal
nR heterozygotic normal
RR homozygotic defective

Albino
Microphthalmos, Anophthalmos
Retinitis Pigmentosa
Myopia
Optic Atrophy
Ectopia Lentis
Glioma Retinæ
Retinal Detachment
Amaurotic Family Idiocy
Day Blindness

3. *Sex-linked Heredity*



xx carrier female normal
xx defective female
xY defective male
xY normal male

Microphthalmos
Anophthalmos
Optic Atrophy (Leber)
Nystagmus
Myopia
Red-Green Colour Blind
Hæmophilia
Macula Absence, Atrophy